Pigs weaned from the sow at 10 days of age respond to dietary energy source of manufactured liquid diets and exogenous porcine somatotropin[1,2]

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ABSTRACT: Previous research indicates that the neonatal pig does not alter feed intake in response to changes in the energy density of manufactured liquid diets. Also, the limited response of IGF-I to exogenous porcine ST (pST) previously observed in young pigs may be influenced by the source of dietary energy. Our objectives were to 1) determine the effect of a high-fat (HF; 25% fat and 4,639 kcal/kg ME; DM basis) or low-fat (LF; 2% fat and 3,481 kcal/kg ME; DM basis) manufactured liquid diet on pig performance; and 2) determine whether the limited response to exogenous pST in young pigs depends on the source of dietary energy. Two replicates of 60 pigs (n = 120; barrows and gilts distributed evenly), with an initial BW of 4,207 ± 51 g, were weaned from the sow at 10 d of age and used in a randomized complete block design. Pigs were assigned by BW to one of six pens. Diets were formulated to provide a constant lysine:ME ratio and were fed on a pen basis for a duration of 9 d. On d 5, barrows and gilts within a pen were assigned randomly to receive either 0 or 120 μg of pST·kg BW^{-1}·d^{-1} for 4 d. Pigs gained 336 ± 9 g/d, which resulted in an ending BW of 7,228 ± 120 g, regardless of dietary treatment ($P > 0.15$). Pigs fed the LF diet consumed 17% more DM per pen daily than pigs fed the HF diet (2,777 ± 67 vs. 2,376 ± 67 g/d, $P < 0.01$), but calculated ME intake did not differ between dietary treatments ($P > 0.20$). The G:F was 24% greater in HF- than in LF-fed pigs ($P < 0.01$). Plasma urea N concentrations were higher in the HF-fed pigs (11.0 ± 0.6 mg/dL) than in pigs fed the LF diet (6.2 ± 0.6 mg/dL; $P < 0.05$). Treatment with pST increased circulating IGF-I ($P < 0.01$) and decreased PUN ($P < 0.01$) concentration 32 and 25%, respectively, regardless of dietary treatment ($P > 0.30$). Circulating leptin averaged 1.8 ± 0.1 ng/mL and was not affected by dietary treatment ($P > 0.35$) or pST ($P > 0.40$). These results suggest that the ST/IGF axis is responsive in the young pig and the increase in circulating IGF-I and growth is independent of the source of dietary energy. Also, young pigs respond to a lower energy density liquid diet with increased feed intake, without altering growth performance, apparently utilizing a mechanism other than circulating leptin.

Key Words: Energy Source, Insulin-Like Growth Factor-I, Leptin, Somatotropin, Swine

 J. Anim. Sci. 2005. 83:1002–1009

Introduction

Improvements through selection, management practices, and nutrition have resulted in dramatic increases in postweaning growth performance of swine, but growth rates have not been improved during the nursing phase. Increased weaning weight decreases postweaning mortality and growth lag, improves nursery performance, and ultimately decreases the age at market weight (Harrell et al., 1993; Kim et al., 2001). Supplemental feeding strategies of nursing pigs provide evidence that the lactating sow does not optimize baby pig growth (Kelly et al., 1990; Azain et al., 1996). In addition, results from artificial rearing studies have indicated that sow's milk does not supply an optimal

[1]We acknowledge O. Phillips and G. Elias for technical assistance.
[2]Presented in part at the Fed. Am. Soc. Exp. Biol., New Orleans, LA, April 20 to 24, 2002, and the joint annual meeting of the Am. Soc. Anim. Sci., Am. Dairy Sci. Assoc., and Canadian Soc. Anim. Sci., Quebec City, Canada, July 20 to 25, 2002.
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Received June 3, 2003.
Accepted January 12, 2005.

pattern of nutrients for neonatal pigs (Auldist et al., 1997). For example, 21-d weights of over 9.5 kg are caused by feeding manufactured liquid diets (Harrell et al., 1993; Oliver et al., 2002). In addition, fat provides approximately 50% of the total calories in sow's milk (Klobasa et al., 1987), which suggests that young pigs require a high amount of dietary fat.

Porcine ST (**pST**) has been utilized effectively to alter the partitioning of nutrients away from fat and toward muscle growth. The administration of pST to growing pigs increased ADG by up to 30%, increased muscle deposition rate by up to 50%, and decreased fat deposition rate by up to 30% (Etherton et al., 1986; Campbell et al., 1988; Dunshea et al., 2002). The response to pST depends on adequate nutrient intake (Campbell et al., 1991; Krick et al., 1993) and stage of development (Harrell et al., 1999). High dietary fat intake may limit the response to pST, as indicated by a greater IGF-I response to ST in humans on a high- vs. low-carbohydrate diet (Snyder et al., 1989). Our objective was to determine whether young pigs alter feed intake in response to dietary energy source (lipid vs. carbohydrate) and whether dietary energy source affects the response to pST.

Materials and Methods

Experiment 1

Animal Care and Dietary Treatments. All procedures were approved by the Institutional Animal Care and Use Committee of North Carolina State University. Two replicates of 60 pigs (n = 120; barrows and gilts distributed evenly) were weaned from the sow at 10 d of age and used in a randomized complete block design. Pigs were blocked by BW and assigned to one of six pens (10 pigs per pen). Pigs were housed in a specialized nursery building (Intensive Care Nurseries, Inc., Colfax, IL) with raised pens, with half of each pen containing an enclosed hover maintained at approximately 32°C, as described by Heo et al. (1999). Ambient temperature was maintained at approximately 24°C. Each block was assigned randomly to either a high-fat (**HF**; 25% fat) or low-fat (**LF**; 2% fat) diet (DM basis). Diets were reconstituted to 150 g/L of water (approximately 12% DM) and were formulated such that the supply of AA per unit of energy was constant (Table 1). The manufactured liquid diet was delivered via a gravity-flow feeding system similar to that reported by Oliver et al. (2002), with 30-L Nalgene carboys (Fisher Scientific, Pittsburgh, PA) used to accommodate each pen of 10 pigs. New supplies of the manufactured liquid diet were added twice daily (0800 and 2000) to ensure freshness and ad libitum access to diets. Liquid diet was prepared on a daily basis and stored at 4°C. Feed disappearance and growth were measured gravimetrically on a daily basis. All components of the feeding system were cleaned thoroughly before the first feeding (0800) with a liquid chlorinated detergent (DS Liquid, Command, Diversey Corp., Wyandotte, MI).

Table 1. Composition and calculated analysis of the dietary treatments[a]

	Diet	
Component	High fat	Low fat
Ingredients, %		
Nonfat dry milk[b]	45.85	42.00
High fat source[c]	30.10	1.50
Lactose[d]	0.00	39.70
Whey protein concentrate[e]	6.00	4.00
Na caseinate[f]	10.00	6.00
L-Arginine·HCl	0.30	0.22
L-Lysine·HCl	0.20	0.13
Xanthan gum	1.00	1.00
$CaCO_3$	0.53	0.37
Dicalcium phosphate	3.75	2.81
Vitamin premix[g]	0.13	0.13
Mineral premix[h]	0.50	0.50
NaCl	0.88	0.88
$MgSO_4$	0.20	0.20
KCl	0.56	0.56
Calculated analyses[i]		
ME, kcal/kg	4,639	3,481
CP, %	31.15	23.50
Fat, %	24.97	1.89
Lactose, %	25.78	60.61
Lysine, %	2.74	2.05
Ca, %	1.11	0.82
P, %	0.76	0.56
CP, g/Mcal of ME	67.1	67.5
Lysine, g/Mcal of ME	5.9	5.9
Lysine, g/100 g of CP	8.8	8.8
Ca, g/Mcal of ME	2.4	2.4
P, g/Mcal of ME	1.6	1.6
Ca:P	1.5	1.45

[a]Expressed on a DM basis.
[b]Milk Specialties Corp. (Dundee, IL).
[c]A blend of edible lard and fancy tallow (80% fat; Fat Pak 80, Milk Specialties Corp.) containing 0.09% 12:0; 1.24% 14:0; 0.16% 15:0; 20.36% 16:0; 2.55% 16:1; 0.41% 17:0; 0.25% 17:1; 10.10% 18:0; 37.01% 18:1; 8.12% 18:2; 0.08% 18:3; 0.17% 20:0; 1.07% 20:1; 0.08% 20:2; and 0.33% 20:4.
[d]Carl S. Akey, Inc., Lewisburg, OH.
[e]Whey protein concentrate (AMP 80; American Meat Protein Corp., Ames, IA).
[f]International Ingredient Co., St. Louis, MO.
[g]Vitamin premix (Milk Specialties Corp.) contained 33,000,000 IU/kg vitamin A; 6,600,000 IU/kg vitamin D_3; 55,000 IU/kg vitamin E; 257,400 ppm vitamin C; 29,983 ppm D-pantothenic acid; 33,069 ppm niacin; 8,378 mg/kg riboflavin; 5,115 mg/kg menadione; 66 ppm biotin, 44,000 ppm vitamin B_{12}; 2,038 ppm thiamine; 3,996 ppm vitamin B_6; and 2,756 ppm folic acid.
[h]Mineral premix (Milk Specialties Corp.) contained 1.002% Ca; 0.549% P; 0.284% Na; 0.040% Cl; 2.024% K; 0.102% Mg; 20,000 ppm Fe; 200 ppm Co; 1,850 ppm Cu; 400 ppm I; 5,000 ppm Mn; 60 ppm Se; and 23,500 ppm Zn.
[i]Calculated analysis based on analysis provided by companies furnishing product and standard feed tables (NRC, 1998).

Blood Collection and Analyses. On d 5, pigs (barrows and gilts) within a pen were assigned randomly to receive either 0 or 120 μg pST·kg BW^{-1}·d^{-1} (Reporcin solubilized in sterile water; Alpharma Co., Toorak, Australia) for 4 d in the extensor muscles of the neck. Pigs selected to receive the 0 pST treatment were injected with an equal volume of sterile saline. Control and pST-treated pigs were in the same pen; therefore, no assess-

ment of the effect of pST on feed intake, ME intake, and G:F was made. The dose of pST or volume of saline was adjusted daily according to pig weights. Blood samples were collected via jugular venipuncture approximately 18 h after the final injection of exogenous pST and immediately placed on ice. After collection, blood samples were centrifuged at 824 × *g* for 10 min, with plasma collected and frozen at –20°C until further analyses. Plasma was analyzed in duplicate for urea N, IGF-I, NEFA, and leptin concentrations. Plasma urea N (**PUN**) concentrations were determined by the quantitative urease/Berthelot procedure (Sigma Diagnostics, St. Louis, MO) based on methods described by Fawcett and Scott (1960) and Chaney and Marbach (1962). Plasma NEFA concentrations were determined by an enzymatic colorimetric method (Wako Pure Chemical Industries, Ltd., Richmond, VA). The sample mean for PUN pools was 6.75 ± 0.07 mg/dL, and the intraassay CV was 2.0%. The sample mean for NEFA pools was 169.6 ± 2.3 mEq/L, and the intraassay CV was 2.7%.

Plasma IGF-I and leptin concentrations were analyzed by RIA. Plasma IGF-I was dissociated from the IGFBP using the glycyl–glycine extraction method as described by Plaut et al. (1991). For the analysis of IGF-I, recombinant human IGF-I was used for standards (Gropep, Ltd., Adelaide, Australia), as well as the radiolabeled ligand (Amersham Parmacia Biotech U.K. Ltd., Buckinghamshire, England). Anti-human IGF-I polyclonal antiserum (GroPep) was used as the primary antibody and goat anti-rabbit γ-globulin (GroPep) was used as the second antibody. The mean concetrations of IGF-I in sample pools were 222 ± 4 and 335 ± 18 ng/mL, and the intraassay CV were 3.4 and 10.7%, respectively. For the analysis of leptin, recombinant human leptin was used for standards and radiolabeled ligand (Linco Research, Inc., St. Charles, MO). Guinea pig anti-multispecies leptin antibody was used as the primary antibody (Linco Research, Inc.). These reagents were validated for use in the measurement of leptin in porcine serum in our laboratory (Whisnant and Harrell, 2002). The mean concentration of leptin for the sample pool was 3.48 ± 0.04 ng/mL, and the inter- and intraassay CV were 2.4 and 3.7%, respectively.

Experiment 2

Eight barrows with an average BW of 82.0 ± 3.5 kg, an age known to be more responsive to exogenous pST (Harrell et al., 1999), were used to verify the leptin results of the pigs from 10 to 19 d of age. Briefly, pigs were individually housed, surgically fitted with indwelling catheters, and had ad libitum access to feed and water at all times. After a 1-wk adjustment period postsurgery, pigs were bled at 8-h intervals for a 48-h period to establish baseline values. Pigs were then injected with 120 μg of pST·kg BW^{-1}·d^{-1} for 4 d in the extensor muscles of the neck. After the initial injection, blood samples were collected every 2 h for 16 h. Blood samples were then collected every 4 h from 16 to 40 h, and every 8 h from 40 to 96 h. Blood samples were processed and analyzed for leptin as described above.

Statistical Analyses. Data were subjected to analysis of variance using the GLM procedure of SAS (SAS Inst. Inc., Cary, NC). Data were evaluated for the effects of energy source, pST, replication, and interactions. The energy source (fat vs. carbohydrate) and pST (0 vs. 120 μg of pST·kg BW^{-1}·d^{-1}) response were contrasted using a protected LSD test (Steel et al., 1997). For live weight, ADG, and all blood measurements, the experimental unit for all statistical procedures was the individual pig. For evaluation of the effects of energy source on ADFI, ME intake, and feed efficiency, the experimental unit was pen of pigs. The significance level for all tests was set at $P < 0.05$.

Results

Pigs gained 336 ± 9 g/d, which resulted in an ending BW of 7,228 ± 120 g (Table 2), regardless of dietary treatment ($P > 0.15$). Pigs consuming the LF diet had 17% greater (2,777 ± 67 DM·pen^{-1}·d^{-1}; Table 2) ADFI than did those consuming the HF diet (2,376 ± 67 g of DM·pen^{-1}·d^{-1}; $P < 0.01$). Estimated ME intake did not differ (Table 2; $P > 0.20$) between diets. As a result of the changes in feed intake and no changes in ADG, G:F was increased by 24% in pigs fed HF compared with LF diets (Table 2; $P < 0.01$). Pigs that received exogenous pST gained weight faster than pigs that did not receive pST (Table 3; $P < 0.05$), regardless of dietary treatment ($P > 0.55$).

Dietary treatment had no effect on basal circulating IGF-I concentrations (Table 3; $P > 0.49$); however, circulating IGF-I concentrations were increased ($P < 0.01$) by 32% in pigs treated with pST, regardless of dietary treatment ($P > 0.40$). Plasma urea N concentrations were 42% lower in the LF-fed pigs than in the HF-fed pigs (Table 3; $P < 0.001$). In addition, exogenous pST decreased PUN concentrations by 25% in both LF- and HF-fed pigs ($P < 0.02$). Plasma NEFA concentrations were 56% less in pigs consuming the LF diet than in pigs fed the HF diet (Table 3; $P < 0.001$). Plasma NEFA concentrations were not affected by pST ($P > 0.45$). Circulating leptin concentrations (Table 3) averaged 1.8 ± 0.1 ng/mL and were not affected by dietary treatment ($P > 0.45$) or exogenous pST ($P > 0.35$).

Discussion

The most convincing data that preweaning pig growth is not maximized by the lactating sow are results from artificial rearing studies (Harrell et al., 1993; Oliver et al., 2002). Harrell et al. (1993) found that artificially reared pigs were 53% heavier at 21 d of age than were pigs reared on the sow (9.8 vs. 6.4 ± 0.5 kg), and the increased preweaning growth resulted in 10 d less time to reach 110 kg BW. Similar 21-d weights were found by Oliver et al. (2002), who examined corn

Table 2. Performance by young pigs fed a high (25%) or low (2%) fat manufactured liquid diet from d 10 to 19 of age[a]

Variable	Diet		SEM	*P*-value for energy source
	High fat	Low fat		
Live weight, g				
d 10	4,210	4,212	96	0.98
d 15	5,656	5,669	111	0.93
d 19	7,270	7,185	122	0.64
ADG, g/d				
d 10 to 15	289	291	10	0.91
d 15 to 19	403	380	11	0.22
d 10 to 19	340	332	9	0.45
ADFI, g DM·pen^{-1}·d^{-1}				
d 10 to 15	1,904	2,242	84	<0.01
d 15 to 19	2,965	3,447	70	<0.01
d 10 to 19	2,376	2,777	87	<0.01
ME intake, Mcal·pen^{-1}·d^{-1}				
d 10 to 15	8.8	7.8	0.4	>0.15
d 15 to 19	13.7	12.0	0.5	>0.10
d 10 to 19	11.0	9.7	0.5	>0.11
G:F (g/g DM)				
d 10 to 15	1.53	1.29	0.02	<0.001
d 15 to 19	1.36	1.08	0.03	<0.001
d 10 to 19	1.44	1.16	0.02	<0.001

[a]Values are least squares means; n = 6.

syrup solids as a replacement for lactose in manufactured liquid diets in pigs from 2 to 21 d of age. In the current experiment, overall ADG was 336 ± 8 g/d. This level of performance is consistent with previous research with manufactured liquid diets (Newport 1979; Heo et al., 1999) in that pigs in the current study had superior performance compared with pigs typically reared on the sow (250 g/d; NRC, 1998). Hence, increased growth by pigs reared independent of the sow could be accomplished with increased supply and/or improved nutrient profile of manufactured liquid diet.

Extensive research has been conducted to optimize the supply of nutrients to maximize lean tissue gain during the postweaning phases of growth, but little has been done in the nursing phases through either changes in sow milk composition or manufactured liquid diets. Fat provides approximately 50% of the total calories in sow's milk (Klobasa et al., 1987), which suggests that the young pig (i.e., <18 d of age) requires a high amount of dietary fat to maximize their BW gains. However, pigs fed the HF or LF diets throughout the current study gained at similar rates (340 ± 9 and 332 ± 9 g/d, respectively), resulting in ending BW of 7,270 ± 119 and 7,185 ± 122 g, respectively. Similarly, Cline et al. (1977) observed no difference in growth rates of pigs weaned at 3 wk of age and limit-fed isocaloric high- or low-fat liquid diets for 2 wk. These data indicate that young pigs are capable of using either fat or carbohydrate equally well as the primary energy source for growth.

Table 3. Effects of exogenous porcine somatotropin (pST) and dietary energy source on ADG and plasma variables of pigs fed a high (25%) or low (2%) fat manufactured liquid diet from d 10 to 19 of age[a,b]

Variable	High fat		Low fat		SEM	*P*-value		
	−pST	+pST	−pST	+pST		ES[c]	pST	ESxpST
ADG d 15 to 19, g/d	393	421	353	405	20	>0.10	<0.05	0.55
PUN, mg/dL[d]	10.9	9.2	6.8	5.0	0.7	<0.001	<0.02	0.75
IGF-I, ng/mL	287	359	252	342	26	0.31	<0.003	0.73
NEFA, μEq/L	190	200	80	44	19	<0.001	0.48	0.22
Leptin, ng/mL	1.8	1.7	1.8	1.8	0.1	0.52	0.48	0.88

[a]Values are least squares means; n = 6.
[b]Injections of 0 or 120 μg of pST·kg BW^{-1}·d^{-1} for 4 d began on d 15 of age. Blood was collected approximately 18 h after the last pST injection.
[c]ES = energy source (fat vs. carbohydrate).
[d]PUN = plasma urea N.

In growing pigs, increased dietary energy concentration resulted in decreased feed intake, whereas ME energy intake remained relatively constant (NRC, 1987). In addition, supplemental fat (10%) decreased feed intake in nursery pigs (Cera et al., 1990; Li et al., 1990); however, Le Dividich et al. (1997) concluded that 1-d-old pigs did not respond to colostral energy concentration with increased feed intake. Although pigs were not allowed ad libitum consumption of the diet, these results may still be accurate due to the limited gastric capacity of pigs at 1 d of age. In the current experiment, 10-d-old pigs that received the LF diet ad libitum consumed 17% more feed than HF-fed pigs. Due to the differences in feed intake with no change in growth rate, G:F was 24% greater in HF- than in LF-fed pigs.

Leptin is involved in the regulation of feed intake and energy balance in a variety of species, including rodents and humans (Ahima and Flier, 2000). Leptin is secreted by adipose tissue and travels via the circulation bound to a specific binding protein, which may modulate its activity, to the brain, where it inhibits the release of the neurotransmitter, neuropeptide Y (Houseknecht et al., 1998). Neuropeptide Y is a potent stimulator of feed intake and inhibits thermogenesis. Therefore, the changes in feed intake in response to energy density observed in the present study could be mediated through leptin. Nonetheless, circulating leptin was unaffected by dietary energy source in the current experiment, despite differences in the level of feed intake and similar ME intakes. In contrast, high-fat diets decreased circulating leptin in both rats (Ainslie et al., 2000) and humans (Romon et al., 1999). Moreover, Cha et al. (2000) observed that high-fat diets (44 or 60%) abolished the diurnal variation of leptin normally observed in rats.

Circulating leptin also was unaffected by exogenous pST in pigs in the present study; however, in older pigs allowed to consume feed ad libitum (Exp. 2), exogenous pST administration transiently increased circulating leptin concentrations by 2 h after treatment ($P < 0.05$) and returned to baseline by 6 h after treatment (Figure 1). Circulating concentrations of leptin were lower at 12, 24, 48, and 56 h after the initial pST injection compared with baseline values ($P < 0.10$). In addition, no diurnal pattern was observed when pigs were bled at hourly intervals for 24 h (data not shown). Due to the transient increase of the initial circulating leptin response to exogenous pST, and inconsistent response after later injections (>6 h), changes in leptin may not have been observed due to the time of blood sampling. Elimam et al. (2001) and Matsuoka et al. (1999) both observed decreased circulating leptin in children in response to ST administration; however, these children were either severely obese (Elimam et al., 2001) or had decreased ST secretory capacities (Matsuoka et al., 1999). Matsuoka et al. (1999) observed that leptin concentrations and adipose mass decreased proportionally, but Elimam et al. (2001) observed a disproportionately greater decrease in leptin compared with fat mass, indicating that ST had effects that were independent of the changes in total body adipose mass. In addition, Spurlock et al. (1998) observed lower leptin mRNA abundance in pigs weighing 30 to 35 kg BW treated with a single 2-mg dose of pST. In contrast, Iglesias et al. (2002) observed increased leptin concentrations after 2 wk of ST treatment in dialysis patients with already elevated levels of leptin due to chronic renal disease.

Plasma urea N concentration is an indirect measure of the extent of AA oxidation, and in the young growing animal that is actively accreting skeletal muscle, it is a measure of the oxidation of dietary AA. Circulating NEFA is an indirect measure of lipolysis and/or fatty acids available for uptake into tissues. In the current study, levels of lipolysis should be very low in pigs weaned from the sow at 10 d of age. Plasma urea N and NEFA were both higher in pigs consuming the HF diet compared with pigs fed the LF diet. Plasma urea N concentrations were approximately 32% less in LF-fed pigs than in HF-fed pigs. Due to similar ME intakes between diets in the current experiment, pigs fed the HF and LF diets consumed similar amounts of CP because the diets were formulated to have a constant CP:ME. Therefore, these data suggest that pigs fed HF diets were oxidizing more dietary AA than LF-fed pigs, and indicate that HF pigs were accruing less muscle, and therefore more fat, than pigs consuming the LF diet. Similar to the results with PUN, circulating NEFA concentrations were approximately 56% less in pigs fed the LF compared with the HF diet. This result was expected because pigs fed the HF diet would have more dietary fatty acids available for tissue uptake. In addition, Tikofsky et al. (2001) observed that bull calves fed a low-fat (14.8%) diet had higher empty body protein and lower empty body fat than calves that consumed a similar amount of energy from a high-fat (30.6%) milk replacer. Although we did not measure effects on body composition, these data suggest the efficiency for AA use for protein accretion is higher in pigs consuming a LF diet than a HF diet. Future experiments are required to confirm the effect of energy source for the young pig on body composition at this stage of development, as well as any changes that are maintained in subsequent phases of growth.

The effects of pST on the neonatal pig have received relatively little attention compared with their more mature counterparts, and the results obtained in young pigs have been inconsistent. Harrell et al. (1999) removed pigs from the sow at 2 to 3 d of age and challenged pigs with 120 μg of pST/kg BW daily for 4 d at 10, 19, 33, 43, 63, 83, and 125 d of age. Differences in response to pST at 10 and 19 d of age were observed in PUN, IGF-I, IGFBP-2, and IGFBP-3, albeit to a lesser extent than in heavier pigs. Similar to the results of Harrell et al. (1999), the increase in IGF-I and decrease in PUN in the current study indicates that the ST/IGF axis is active in the young pig. In addition to changes in metabolic signals, we observed a growth response; ADG was increased approximately 11% in pigs treated with

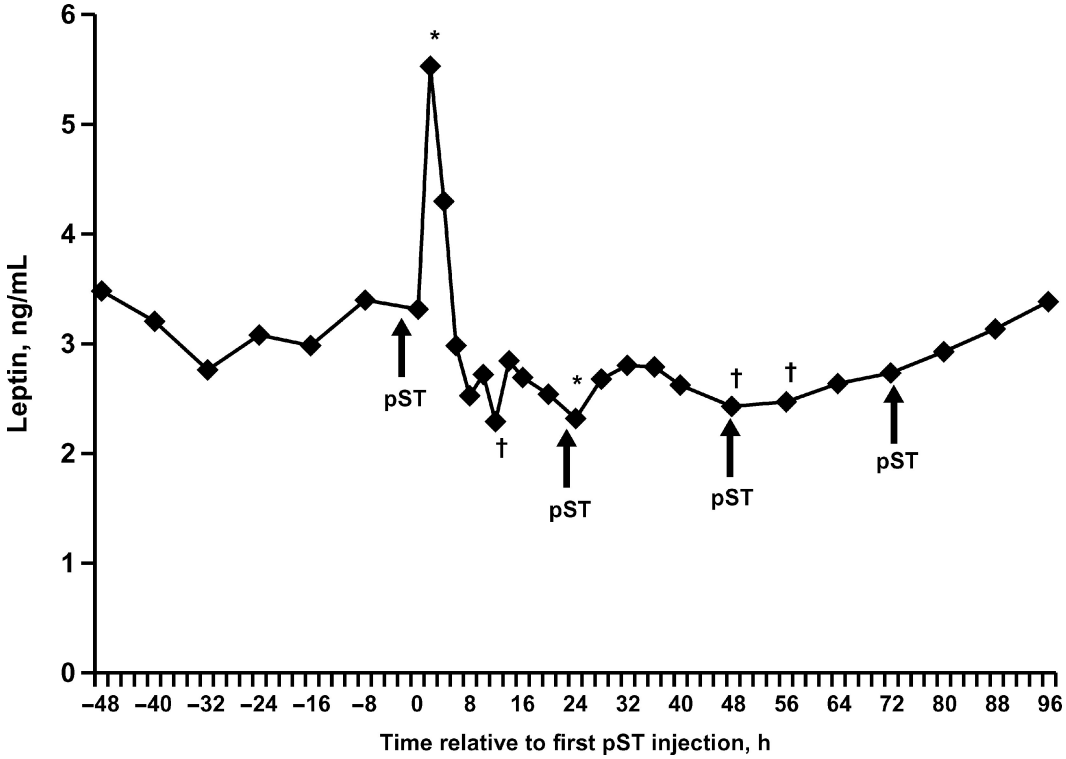

Figure 1. Effect of porcine somatotropin injection (pST) on circulating leptin concentrations in pigs weighing 82.0 ± 3.5 kg and allowed to consume feed ad libitum. Pigs (n = 8) were surgically fitted with indwelling jugular catheters and received 120 μg of pST·kg BW^{-1}·d^{-1} for 4 d after a 1-wk adjustment period. Blood samples were collected every 8 h for 48 h before pST injections to establish baseline values. Blood samples were collected every two hours for 16 h beginning immediately after the first pST injection. Blood samples were then taken every 4 h from 16 to 40 h after pST treatment and every 8 h from 40 to 96 h after pST treatment. Values shown are means. Pooled SEM = 0.11. †pST effect, $P < 0.10$; *pST effect, $P < 0.05$.

exogenous pST. However, we did not observe a difference in circulating NEFA due to exogenous pST, as was found in older pigs (Wray-Cahen et al., 1991; Dunshea et al., 1992). Wester et al. (1998) also observed changes in IGF-I, IGFBP-2, and IGFBP-3 in response to pST in pigs weaned from the sow at 12 h to 1 d of age and fed manufactured liquid diets. In addition, BW for pigs that received pST were heavier after the 7-d experiment. However, the levels of pST administered in the study of Wester et al. (1998) were 10- to 15-fold higher (1 mg/kg BW given in three equal injections per day) than the more common dose of 120 μg of pST·kg BW^{-1}·d^{-1}, which maximized lean tissue deposition in growing pigs from 30 to 90 kg BW (Krick et al., 1992). In contrast, no differences were observed by Dunshea et al. (1999) in growth performance or circulating concentrations of IGF-I, IGF-II, or IGFBP-3 when daily injections of pST (60 μg/kg BW) were given to nursing pigs from d 4 to 31 of lactation. Also, Dunshea et al. (2003) failed to see an increase in growth rate of sow-reared pigs receiving daily injections of pST at higher doses (1 mg/kg BW). At growth rates typical of pigs reared on the sow, the restraint of growth leading from a restricting amount of nutrients available from the sow could cause an attenuated response to pST. For example, when the effects of exogenous pST were titrated by different protein levels (8.3 to 23.4 g/100 g of feed, as-fed basis) the expected effects on growth performance and protein deposition were observed with the four highest protein diets, but not the two lowest protein diets (Campbell et al., 1990). In addition, circulating concentrations of IGF-I and IGFBP-3 were decreased, and concentrations of IGFBP-2 were elevated in conditions of poor nutritional status (Clemmons and Underwood, 1991; Thissen et al., 1994; Underwood et al., 1994), which indicated a decreased responsiveness of the ST/IGF axis. In contrast to the results of Dunshea et al. (1999), it was observed by Harrell et al. (1999), as well as in the current experiment, that the somatotropic axis is functional in pigs consuming manufactured liquid diets ad libitum and growing at rates superior to those of pigs reared on the sow.

In the current experiment, the response to pST did not differ between diets, indicating that the source of dietary energy did not affect the response to exogenous pST. In contrast, Snyder et al. (1989) observed lower urinary N losses and a greater IGF-I response to exogenous ST (0.1 mg/kg initial BW every other day) in human subjects consuming 72% of their nonprotein calories as carbohydrate compared with subjects consuming

80% of their nonprotein calories as lipid; however, these subjects were in a negative energy balance. Similar to the results of the current experiment, Azain et al. (1992) observed no differences in response to pST in pigs consuming a diet with 10% supplemental fat compared with a diet with no supplemental fat, although the pigs were older and heavier (87 kg) than in the present study. These data, along with the current experiment, suggest that the responsiveness of the ST/IGF axis is not affected by dietary energy source.

This study confirms earlier data that the nutrient pattern of sow's milk may not be optimal for maximizing pig growth. For example, growth performance of pigs was maximized when the supply of lysine per unit of energy was approximately 50% greater than that found in sow's milk (Auldist et al., 1997). Furthermore, diets that were used in artificial rearing studies, including the current study, supplied approximately 50% greater AA content per unit of energy than sow's milk and resulted in greater pig weight gains (Harrell et al., 1993; Oliver et al., 2002). In addition, supplementing lactating sow diets with CLA decreased sow milk fat content by approximately 35%, but growth performance of the nursing litters was not altered (Harrell et al., 2000). Results of the present study suggest that the ST/IGF axis is active in the young pig, but that it is not influenced by the source of dietary energy.

Implications

The results from this experiment clearly show that the young pig (10 d of age) responds to the energy density of the diet. Pigs that received low-fat vs. high-fat diets had increased feed intake, but similar metabolizable energy intake and rates of growth. In addition, the somatotropin/insulin-like growth factor axis seems to be responsive in the young pig, and the increase in insulin-like growth factor-I and growth with exogenous porcine somatotropin was independent of the dietary energy source. The differences in feed intake to obtain similar levels of metabolizable energy intake utilize a different mechanism than circulating leptin. These results indicate that feed manufacturers could alter dietary formulations for early-weaned pigs fed manufactured liquid diets, depending on the availability and economics of dietary ingredients.

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